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THE EFFECTS OF SLEEP DEPRIVATION AND EXERCISE ON HEART RATE DURING RECOVERY SLEEP

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Abstract

Based on recent findings that sleep following exercise is sometimes characterized by more slow-wave sleep (SWS) and higher heart rate (HR) levels compared to sleep not following exercise, it has been argued that exercise depletes bodily resources, and these cortical and autonomic changes reflect the need for enhanced restitution during sleep. Prolonged sleep loss produces similar cortical and autonomic effects. The study reported here investigated whether sleep loss or exercise would independently increase HR during the next available ("recovery") sleep period, and whether sleep loss plus exercise would have a synergistic effect on HR. HR was measured during baseline sleep and during recovery sleep following each of three conditions of energy depletion: a) exercise with regular sleep, b) 65 hr of sleep loss with exercise, and c) 65 hr of sleep loss without exercise. In the exercise conditions, the exercise consisted of treadmill running, ergometer cycling, anaerobic tests, tests of isokinetic strength and endurance, and tests of isometric force and reaction-time on the first and third days, and a 6 hour hike at 24% of the subjects' maximal aerobic capacity on the intervening day. In contrast to HR levels during the baseline sleep, HR was higher during the recovery sleep for the sleep loss and sleep loss plus exercise conditions, but not for the exercise condition with regular sleep. Furthermore, the HR levels during the sleep loss plus exercise condition did not exceed the HR levels following the condition of sleep loss alone. These results suggest that prolonged sleep loss is sufficient to raise HR levels during recovery sleep. However, exercise did not cause similar elevations in HR during recovery sleep nor did exercise augment the sleep loss effect. These data suggest that the neural regulation of the heart and the normal autonomic balance during sleep is altered when the need for restitution during sleep is enhanced by prolonged wakefulness.

Introduction

Sleep is a necessary requirement for normal human functioning, but the reason why we sleep is not known. One theory is that sleep serves to facilitate restoration wherein body tissues are repaired or returned to normal levels of functioning, and cerebral processes are recuperated. Experimental investigations of the role of sleep in restoring physical and/or mental capabilities typically involve manipulating subjects' level of physical activity and/or their amount of sleep. Exercise and sleep deprivation both lead to a heightened need for homeostatic restoration due to an exaggerated depletion of resources compared to normal activity and wakefulness.

There exists a considerable literature relating the effects of exercise and sleep deprivation to subsequent sleep. The primary finding of this exercise literature is that the amount of slow wave sleep (SWS), consisting of sleep stages 3 and 4 (1), increases with increasing fatigue induced by daytime exercise (2,3), though perhaps only in physically fit subjects who engage in habitual exercise (2, 4-6). Also, it has been well substantiated that as little as 40 hr of sleep deprivation results in an increase in the amount of SWS, which has been argued to be a compensatory rebound due to SWS deprivation (3). The interactive effect of total sleep deprivation and exercise on SWS has also been reported by Naitoh, Englund, Ryman and Hodgdon (7). In their study fit subjects were assigned to one of two experimental conditions: 45 hr of sleep loss with or without exercise (treadmill walking). Not only was SWS enhanced following sleep loss during the next available (here termed "recovery") sleep period, but exercise potentiated the impact of sleep loss on SWS. The findings that exercise and sleep loss increase SWS during recovery sleep provides support for the notion that SWS is related to the restoration of physiological states (3, 8-11).

The interpretation that increased SWS is correlated with greater restitution would be more credible if changes in SWS were accompanied by other physiological changes consistent with an enhanced need for restitutional processes. One physiological system that would be a good initial candidate for alterations during recovery sleep would be the cardiovascular system. If an increase in SWS is one facet of increased restitutional processes, then one could anticipate changes in autonomic functions, such as heart rate (HR), during recovery sleep, since the primary role of the cardiovascular system is to support altered metabolic requirements.

While changes in HR levels during normal sleep have been examined quite extensively (12-18), few studies have examined changes in HR during sleep after either exercise or sleep loss. In terms of exercise, the general finding has been that, compared to HR levels during a baseline sleep, HR is elevated during recovery sleep following prolonged exercise. For example, Hauri (19, 20) reported that, following 6 hours of exercise, HR was significantly elevated during the first three hours of sleep. Desjardins, Healey and Broughton (21) found HR to be elevated up to 60 min after bedtime following different intensities of treadmill exercise, but HR did not increase proportionally with increasing intensities of exercise, possibly owing to a ceiling effect. Walker et al. (22) reported HR elevations in recovery sleep after distance running in those subjects who were unaccustomed to running, but not in trained distance runners. While studying sustained marching, Roussel and Buguet (23) found that during a recovery sleep period following 6 days of 35 km marches, HR was significantly increased (by about 10%) over the HR levels during a baseline sleep. Similarly, Bonnet (24) found that HR was significantly elevated throughout the first night following a 32 km march (24). He also reported that HR was elevated during the first three hours of the second night following the march even though the daytime waking HR levels during the second day were similar to baseline daytime levels.

The influence of exhaustive submaximal exercise (walking on a treadmill) on cardiac activity during sleep was more recently examined by Bunnell, Bevier & Horvath (25). This was the first study to report an elevation in HR accompanied by an increase in sleep depth (i.e., more SWS) following exercise. On the night of the exercise day, HR was significantly higher throughout the entire 6-hour sleep, compared to the baseline night, with the highest HR levels being accompanied by an increase in Stage 4 sleep. This report followed previous studies by Bunnell and his colleagues in which they found that after submaximal exercise, HR was elevated by 5.4 beats/min during sleep on the exercise night compared to the sleep on the baseline night (5). Similarly, Bunnell et al. (26) reported an elevation in HR during the first two hours of sleep following daytime maximal-capacity exercise compared to corresponding HR levels during baseline sleep.

While some studies have not found HR elevation during recovery sleep following exercise (e.g., 27), the general finding is that when the need for restitution is enhanced through exercise, HR appears to be elevated during recovery sleep. Even fewer studies have been reported on HR changes during recovery sleep when the need for restitution is enhanced by the prolonged energy depletion associated with sleep loss.

One recent study examined changes in HR levels during recovery sleep after subjects underwent 64 hr of total sleep deprivation and continuous cognitive work (28). During recovery sleep, HR levels were found to be significantly higher (by about 10%) than during the baseline sleep. In addition, this relative increase in HR was also accompanied by a relative increase in the amount of SWS compared to the baseline sleep. These HR results were replicated in 2 subsequent studies (29, 30). In the first of these studies, the authors reported that during both a 2-hr nap, which followed 40 hrs of sleep loss, and a subsequent recovery sleep after another 22 hrs of wakefulness, HR was significantly increased over the baseline sleep HR levels by about 7%. In the second of these studies the nap was placed after 46 hours of sleep loss and the recovery sleep followed an additional 16 hrs of wakefulness. In that study (30), HR levels were significantly increased over baseline levels by 5% during the nap and by 13% during the recovery sleep. Thus, it appears that the recovery process following sleep loss requires enhanced cardiac activity which occurs in conjunction with enhanced sleep depth (i.e., more SWS).

Since cardiac activity appears to be altered during sleep following both exercise and sleep loss, the present study attempted to examine the independent and combined effects of exercise and total sleep deprivation on cardiac activity during recovery sleep. It was predicted that enhanced restitutional processes would be manifested by an elevation in HR during recovery sleep, such that HR was expected to be higher during recovery sleep than during the baseline sleep following both exercise and sleep loss conditions. Furthermore, because energy depletion and the need for restitution is presumably greater when subjects undergo both exercise and sleep loss than when they undergo either independently, it was predicted that HR during sleep following both exercise and sleep loss would exceed the HR increase following either condition alone.

Method

Subjects were 11 male volunteers averaging 20 years of age. Their mean bicycle maximal aerobic power (VO_2max) was 50.4 ml/kg/min, and their mean treadmill VO_2max was 53.7 ml/kg/min. All subjects participated in each of the following three conditions: a) sleep and exercise (SE), b) 65-hr sleep deprivation and no exercise (sedentary) (SDS) and c) 65-hr sleep deprivation and exercise (SDE). Subjects were run in two groups of four and one group of three (a twelfth subject withdrew from the experiment). Each subject participated in all conditions. The order in which the groups of subjects participated in the 3 conditions was counterbalanced across groups of subjects and each condition was separated by at least 10 days.

In all conditions, on the evening prior to Day 1, subjects slept in the laboratory for six hours (2400-0600h) during which time ECG signals were continuously recorded using ambulatory Oxford Medilog 9000 recorders (baseline measures). When subjects were in the exercise conditions (SE, SDE), physical performance testing was conducted on Day 1 (following the baseline night of sleep) and on Day 3, following either two nights of sleep or two nights of sleep deprivation. These performance tests included ergometer cycling, treadmill running, anaerobic tests, tests of isokinetic strength and endurance, and tests of isometric force and reaction-time. The schedule of daily activities for Days 1 and 3 are shown in Table 1.

TABLE 1. Activity Schedule of Day 1 and Day 3 for Exercise Conditions

TIME	ACTIVITY
0700-1030	20 min on a cycle ergometer at 70 % VO2 max
1030-1100	standard breakfast
1100-1200	tests of isokinetic muscular strength and endurance
1200-1300	cycle intermittent exercise
1300-1400	Wingate Anaerobic Power Test
1400-1500	tests of isometric muscular strength and reaction time
1500-1730	treadmill running at 70% and 80% VO2 max
1730-2030	oral glucose tolerance test
2030-2100	standard dinner
2100-2300	a series of psychological tests
2300	bedtime

On Day 2, subjects in the SE and SDE conditions underwent a six-hour hike (covering approximately 11 miles) in a local park, between 1000 and 1630h (with the exception of a 30-min lunch break). This constituted the major physical activity of the study, and subjects marched at a mean rate of 24% of their VO_2max (31). During this time HR was continuously measured and oxygen consumption was measured for 15 min on three separate occasions. Except for sleep in the SE condition, the regimen for both exercise conditions was identical. In this particular report, only sleep is of interest and other aspects of the study are reported elsewhere (31).

All groups underwent psychological testing at approximately three-hour intervals for 10-20 min from 0600-2000h on Days 1, 3, and 4, and underwent an extended battery of psychological tests from 2100-2300h on Days 1, 2, and 3. In addition, subjects in the SDS and SDE conditions performed psychological tests for 30 min of each hour from 0001-0600h on Days 2 and 3. These subjects occupied themselves at other times by reading, watching movies, conversing, playing games and taking occasional nonvigorous walks. For those in the SDS condition, these activities were conducted during the daytime on Days 1, 2, 3 and 4, as well as overnight. To further ensure wakefulness, subjects were accompanied by two experimenters at all times. On the evening of Day 3, subjects in all conditions underwent a six-hour recovery sleep from 2400-0600h during which time ECG signals were continuously recorded.

To summarize, all subjects were exposed to three experimental conditions (SE, SDE and SDS), and in each condition, HR was measured during baseline and recovery sleep (that followed either two nights of sleep or two nights of sleep deprivation with or without exercise).

Results

The purpose of this study was to determine whether heart rate was higher during recovery sleep than during baseline sleep under three conditions of energy depletion; i.e., SE, SDE, and SDS, and whether HR during recovery sleep in the SDE condition would exceed that in the SE and SDS conditions.

The HR (beats/minute) data were first summarized in 30-sec epochs which were then collapsed into five-minute blocks for subsequent analyses. Data were available for the first three hours (36, five-minute blocks) of sleep for all subjects. Rather than analyzing absolute HR values, analyses were based upon standardized scores calculated as a function of the initial resting levels during the first 5 minutes after subjects went to bed. More specifically, each subject's HR level during each five-minute block of each condition was calculated as a percentage of his resting supine level during the first five minutes following "lights out" with the initial 5-min resting period representing 100%. Standardized scores rather than absolute HR values were used for the following reason. Different subjects participated in the three treatment conditions, and considerable inter-subject variability in HR was found throughout the baseline night and during the initial resting supine levels on both baseline and recovery nights. This type of analysis corrected for individual differences in HR in initial resting levels. Furthermore, when HR scores were transformed to percent change scores, the topography of the HR change during the three baseline night's sleep was identical for all three conditions and, as expected (36), HR decreased during the first 20 min of sleep in all sleep conditions. Finally, since the present study was primarily interested in examining the changes in HR topography that occurred after the initial drop in HR following sleep onset, standardizing the HR levels made it easier to examine the topographical changes following sleep onset.

The study originally consisted of 12 subjects (four subjects per condition) but due to problems with the collection of ECG data and the withdrawal of one subject from the study, only five subjects had complete data across all three experimental conditions. These five subjects were included in a 2(Night) X 3(Condition) X 36(Block) analysis of variance (ANOVA), with Night (baseline versus recovery), Condition (SE, SDE, SDS) and Block (36, five-minute segments) as within-subject factors. The graph showing the change in HR from resting levels over Blocks as a function of Night and Condition appears in Figure 1.

Significant main effects of Condition ($F(2,8)=4.25$, $p=.055$) and Block ($F(35,140)=1.80$, $p<.01$) on HR were qualified by a significant Condition X Block interaction ($F(70, 280)=1.41$, $p<.05$). HR during the recovery sleep in the SDE and SDS conditions was higher than in the recovery night for the SE condition and all baseline sleep HR levels, but HR recovery levels in the SDE and SDS conditions did not differ from one another. All 3 baseline nights were identical for all conditions.

In the SDE and SDS conditions, HR during recovery sleep decreased by 5% within a period of 10 min after "lights out" and gradually increased and maintained a level for the following three hours that was elevated over the baseline level. The recovery sleep for the SE condition resembled that of the baseline night's sleep for all conditions in that HR dropped throughout the first 25 min following "lights out", leading to a 15% decrease for most of the 3-hr period.

Because HR was higher in both sleep deprivation recovery conditions (SDE, SDS) compared to the SE recovery condition, and because a difference was not found between the SDE and SDS conditions, the elevation in HR during recovery sleep could be attributed to the effects of sleep deprivation, but not exercise.

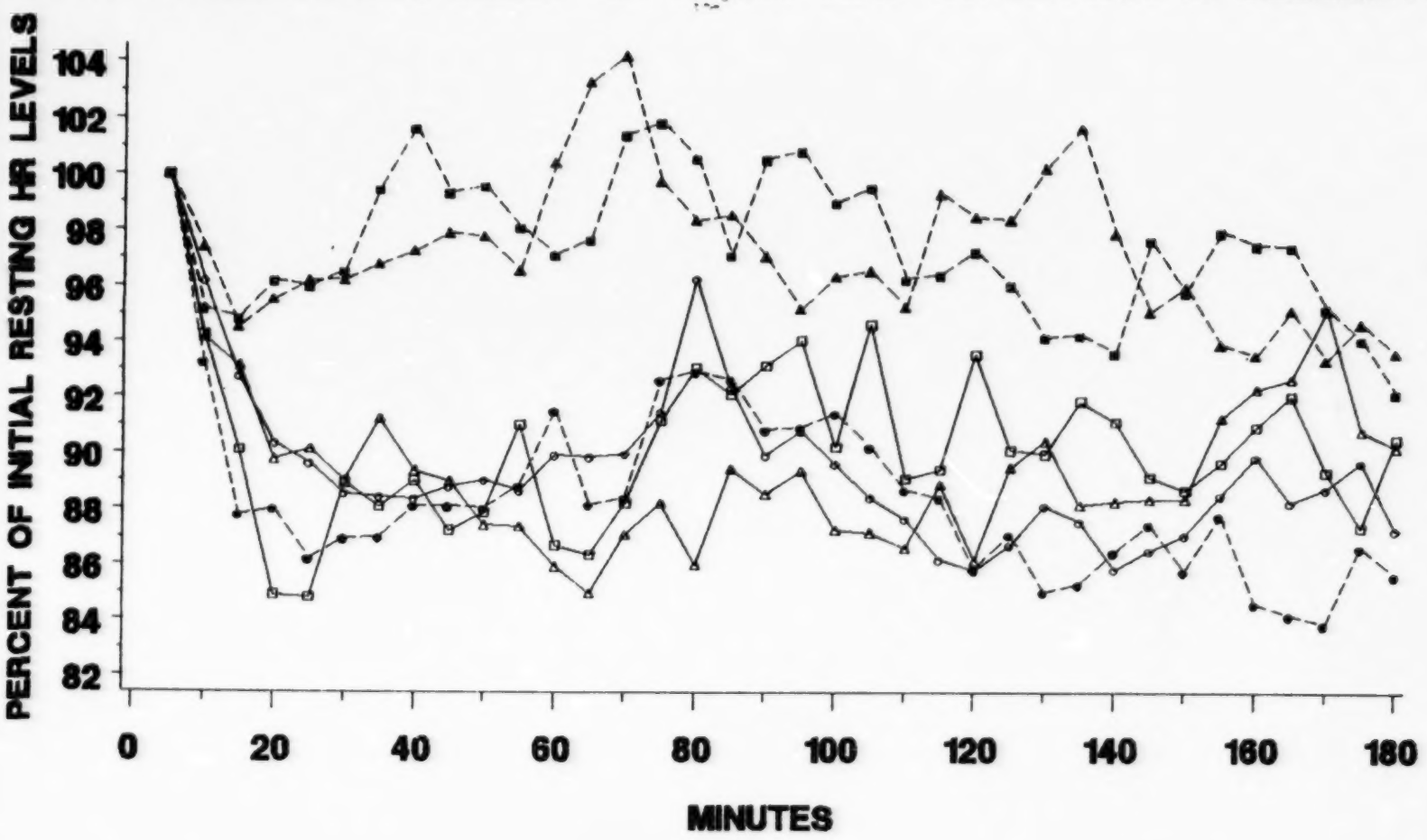
Although only five subjects could be included in the above analysis, a larger sample could

be used to compare HR levels during baseline and recovery sleep for each condition separately, by including subjects who had data for the baseline and recovery nights in each condition. The SE, SDE, and SDS conditions had data available for nine, nine and eight subjects, respectively.

For each condition (SE, SDE, SDS) a 2(Night) X 36(Block) ANOVA was conducted, with Night (baseline versus recovery) and Block (36, five-minute segments) as within-subject factors. Results for each energy-depletion condition are presented separately below. Graphical depictions of the change in HR from resting levels over Blocks, for the baseline and recovery periods, are presented in Figures 2, 3, and 4 for the SE, SDE and SDS conditions, respectively.

FIGURE 1

HR CHANGE DURING SLEEP BY NIGHT AND CONDITION (5 SUBJECTS)



Sleep and Exercise

The only significant effect on HR was a main effect of Block ($E(35, 280)=4.77$, $p<.001$). Following a steep drop coinciding with "lights out", HR maintained a steady level until approximately 75 min into the sleep period. Here, HR increased slightly and then continued to drop throughout most of the remaining 3-hr period. This temporary increase was probably coincidental with the onset of the first REM period but this cannot be confirmed because EEG data were not analyzed.

Sleep Deprivation and Exercise

Significant main effects of Night ($E(1, 8)=6.00$, $p<.05$) and Block ($E(35, 280)=3.95$, $p<.001$) were found for HR. HR scores were significantly higher during recovery sleep compared to baseline sleep. A rapid decline in HR at sleep onset was followed by an elevation in HR for about 90 min after sleep onset on the recovery night. Thereafter, HR progressively declined but remained above baseline levels. The data suggest that HR was primarily elevated over the first sleep cycle.

Sleep Deprivation and Sedentary

Significant main effects of Night ($E(1, 7)=7.10$, $p<.05$) and Block ($E(35, 245)=1.81$, $p<.01$) were qualified by a significant Night X Block interaction ($E(35, 245)=2.56$, $p<.001$). HR was higher during recovery sleep than during baseline sleep, and it dropped by about seven percent during the first 10 min following "lights out" on both nights. HR continued to decline during the next 50 min (12% decline) during baseline sleep, whereas during recovery sleep it gradually increased during the next 60 min. Again, HR during recovery sleep was markedly elevated over baseline sleep.

Thus, the analyses that compared HR during baseline and recovery sleep for each condition separately, yielded results consistent with the former 2 X 3 X 36 ANOVA that consisted of only five subjects. More specifically, HR was higher during recovery sleep than during baseline sleep in the SDE and SDS conditions, whereas this was not the case for the SE condition. These parallel findings strengthen the argument that sleep loss, and not exercise, produced an elevation in HR during recovery sleep.

FIGURE 2

HR CHANGE DURING SLEEP FOLLOWING REGULAR SLEEP AND EXERCISE

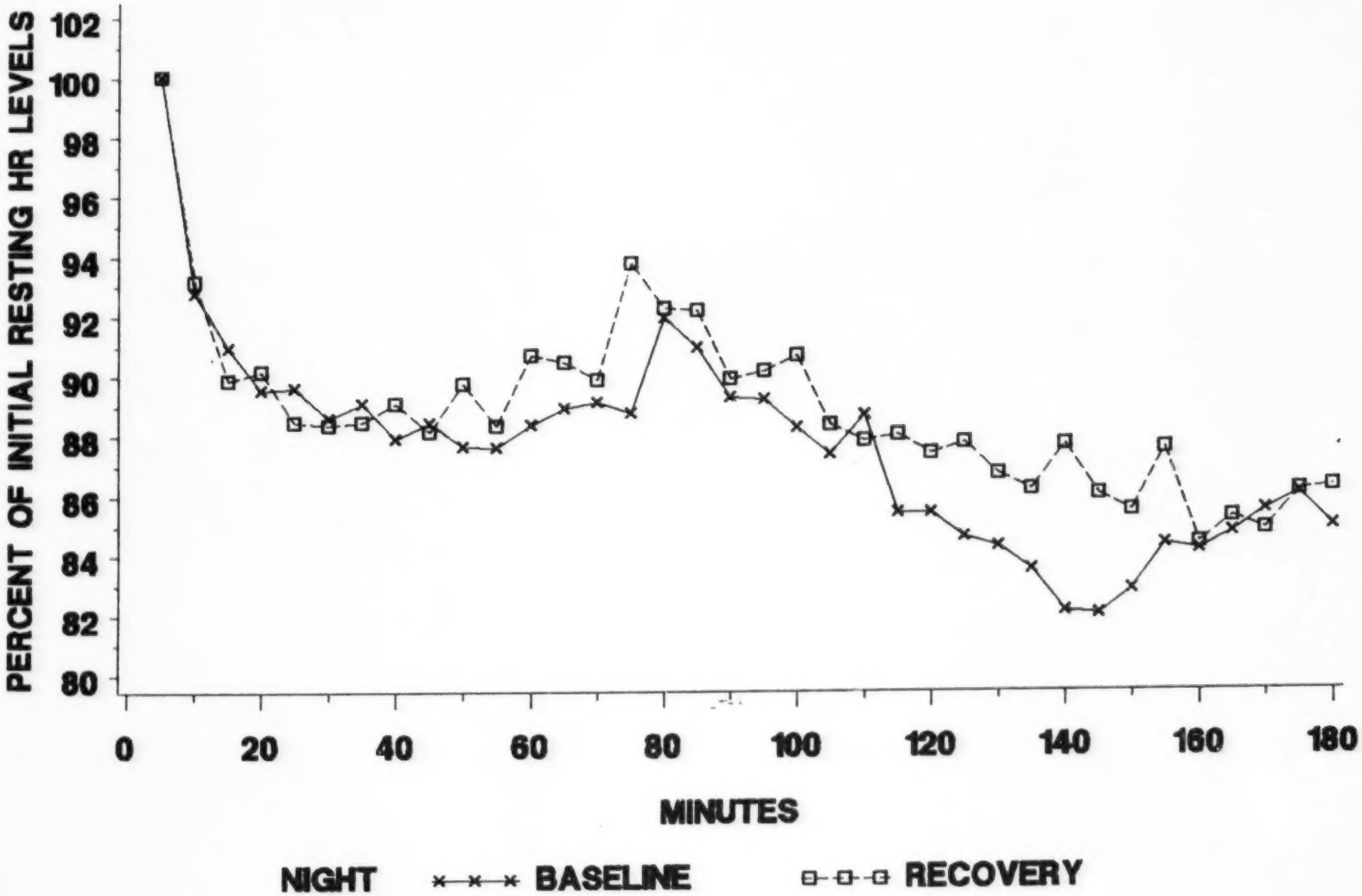


FIGURE 3

HR CHANGE DURING SLEEP FOLLOWING SLEEP LOSS AND EXERCISE

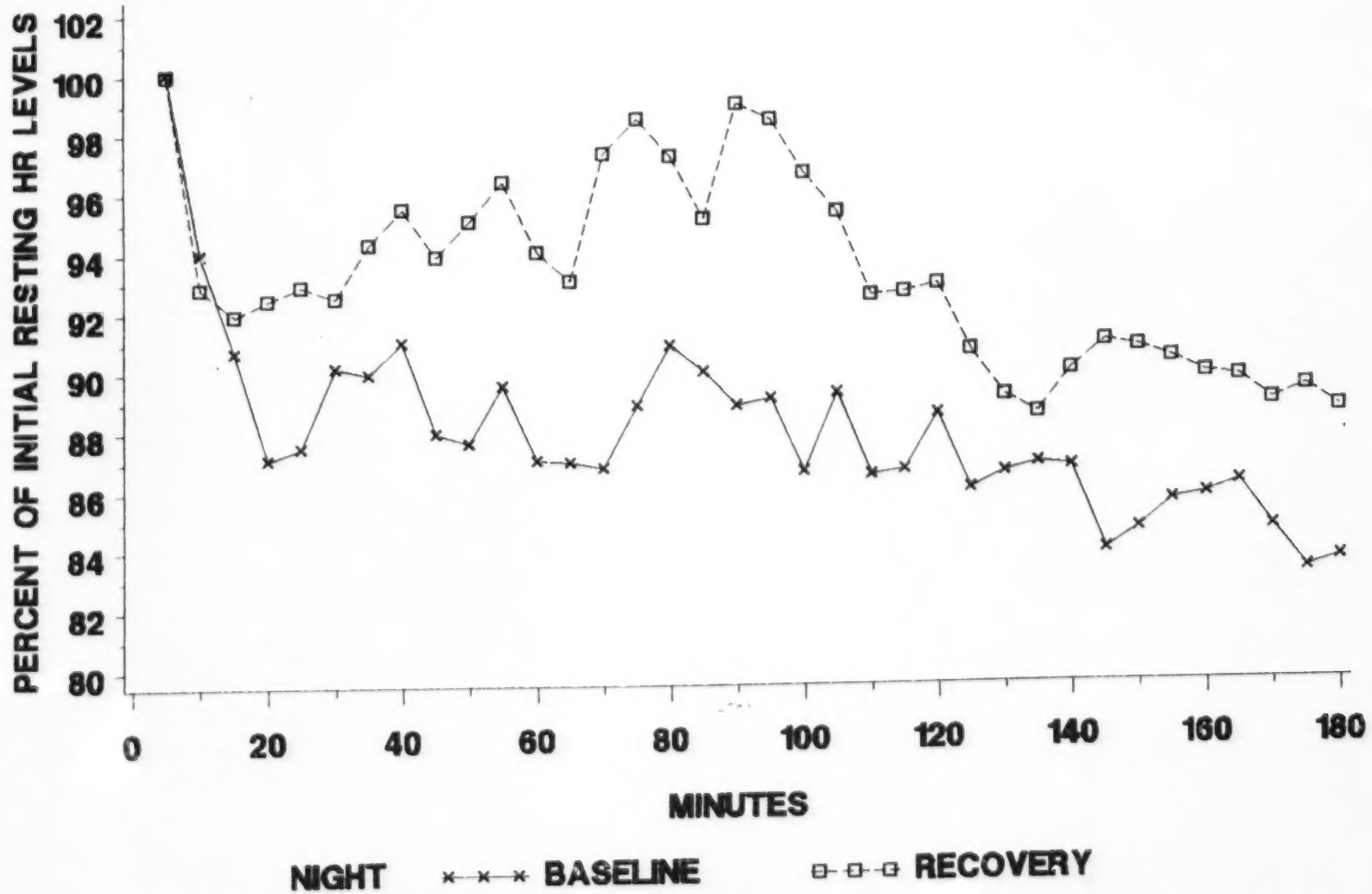
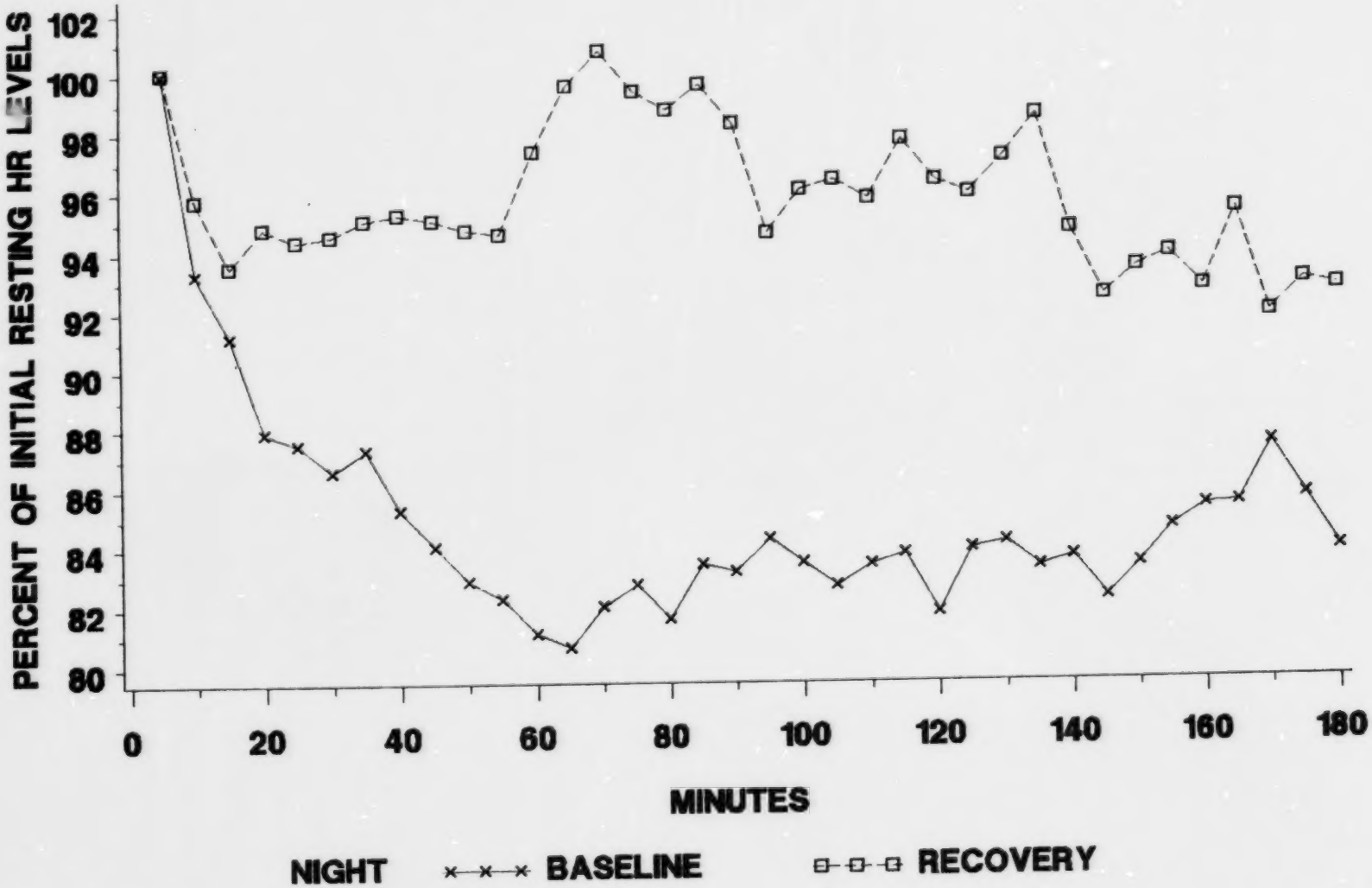


FIGURE 4

HR CHANGE DURING SLEEP FOLLOWING SLEEP LOSS ALONE



Discussion

It was hypothesized that a) exercise and sleep deprivation would increase HR during recovery sleep relative to baseline sleep, and b) HR during recovery sleep in the SDE condition would exceed HR in the SE and SDS conditions because of the additional restitutional requirements imposed by two conditions of energy depletion. The results showed that HR was significantly higher during recovery sleep than during baseline sleep in the SDE and SDS conditions, but this was not the case in the SE condition. The amount of the increase in HR levels during recovery sleep in the SDE and SDS conditions, however, did not differ. Such findings imply that sleep deprivation and not exercise was the major factor contributing to increased HR, relative to baseline sleep characteristics. Thus, the first hypothesis was only partially supported whereas no support was provided for the second.

Increased HR during recovery sleep following sleep loss is in line with earlier studies (28-30) and these data suggest that this elevation in HR levels is a component of the enhanced restoration that is likely occurring during recovery sleep. This conclusion must be considered tentative, however, since other measures of enhanced restitutional processes, such as SWS, were not obtained in this study. In addition, future work in this area would benefit from the inclusion of an activity measure in order to rule out the slight possibility that increased HR was due to an increase in motor activity during recovery sleep. However, this is unlikely since REM sleep is usually delayed and reduced in such recovery sleep. Finally, it would have been interesting to obtain HR data on the night following recovery sleep, in order to determine whether the effect would persist beyond the first recovery night.

Contrary to expectations, and to several earlier studies (5, 19, 20, 22-26, 32), exercise did not increase HR during recovery sleep. Although it is not clear why this effect was absent, a number of plausible explanations can be offered. First, it is possible that this study differed from previous studies that reported elevated HR levels after exercise in some important methodological way. To examine this possibility, a general methodological comparison of this study was made with those studies yielding enhancing effects of exercise on HR during recovery sleep (5, 19-26, 32). It was found that these nine studies had sample sizes ranging from 4 to 20 subjects, employed males in all studies except for three studies that employed both sexes, and had a mean age of either 20 years or 25 years except for one study whose mean age was about 33 years. All studies incorporated a period of laboratory familiarization, but only two studies employed more than one baseline night. Physical activity, typically conducted during the afternoon and/or evening (two studies used morning and afternoon exercise), included marching, ergometer cycling and weightlifting, treadmill running, and cross-country running. However, in studies employing a common activity, the intensity and duration of the activity varied. Furthermore, the criterion for defining fitness was inconsistent across studies.

The only variable in the present study that apparently deviated from the other nine studies as a whole was the type of exercise. With the exception of three studies, all other studies involved some form of running. In contrast, the present study involved a total of 2.5 hr of treadmill running, ergometer cycling, and anaerobic tests such as the Wingate Anaerobic Power Test, tests of isokinetic strength and endurance, tests of isometric force and reaction-time, and a six-hour hike.

It is tempting to conclude that a deviation in exercise type between the present study and the above-mentioned studies may be an important factor accounting for the discrepancy in HR effects. However, such a conclusion can only be valid if it is ascertained that these varied activities are comparable in terms of exercise load. According to Horne (2), the duration of the task and rate of energy expenditure (REE) are two variables necessary for the assessment of exercise load, and they can be combined to provide a measure of total energy expenditure (TEE).

Under the assumption that exercise load is the key factor in determining the need for restorative processes following exercise, it is essential to compare physical activity on the basis of an underlying commonality, i.e., TEE or REE. Otherwise, comparisons across exercise-types may be capitalizing on unknown dimensions. Unfortunately, studies have failed to report data in this manner.

The major physical activity in the present study was the six-hour hike that took place on Day 2. Therefore, it may be instructive to compare this study with the other two studies that employed marching as a physical activity and that found an increase in HR during recovery sleep (23, 24). Bonnet (24) required subjects to march for approximately 32 km on a treadmill between 0745 and 1600 h. On the other hand, Roussel and Buguet (23) required subjects to march at 35% of their VO_2max for 34 km between 0900 and 1700 h on each of six consecutive days. In the present study, subjects engaged in six hours of hiking at a mean rate of 24% of their VO_2max (treadmill-determined). As far as this particular activity was concerned, this study yielded the lowest energy expenditure (although it must be recalled that subjects engaged in a number of performance tests). Thus, one could suggest that the failure to find the anticipated exercise-induced increase in HR, may be due to the fact that the degree of catabolic activity induced by exercise was insufficient to necessitate additional restorative processes during recovery sleep.

With respect to hiking activity, the studies by Bonnet (24) and Roussel and Buguet (23) can also be differentiated from the present study in terms of time elapsed between the exercise and the collection of HR data during sleep. For instance, in those studies HR was first measured during the same night that followed the daytime exercise. The period of time that elapsed between the end of the marching activity and bedtime was about 6 hours in the former study, and 5 hours in the latter study. In contrast, the recovery sleep in the present study did not occur until the second night following hiking in the SDE condition. The 31 hr lag between the hike and the recovery sleep in the SDE condition could account for the lack of an exercise effect although strenuous exercise testing did occur during the third day prior to the recovery sleep. One might also expect performance tests on Days 1 and 3, and hiking on Day 2 to have a cumulative effect leading to an even greater need for restitution during the first night of recovery sleep. (It should also be remembered that Bonnet (24) found HR elevation over baseline levels in his study even during the early portion of the second night of sleep. Given these data one would have expected an exercise effect during the second night of sleep in this study as well.) For the SE condition, the recovery sleep reported was essentially the second night of sleep following the hike. An alternative explanation for the lack of an exercise effect in the SE condition is that there was no sustained exercise effect over two nights but it is possible that there may have been some effect during the sleep on Day 2.

On the other hand, the negative finding of exercise on HR may be at least partially due to the fact that subjects had ample opportunity to physically recover from their exercise during the interim between hiking activity and recovery sleep even though they did not sleep in the SDE condition. In fact, this interpretation is in line with a conclusion drawn by Horne and Porter (33). Because a moderate exercise load incurred early in the day failed to affect SWS during recovery sleep, Horne and Porter (33) concluded that the ensuing wakefulness was sufficient to allow significant recovery. This finding, along with the negative finding of exercise on HR in the present study, might imply that sleep is not necessary for recovery from physical exercise. This interpretation is interesting in that it suggests that restitutional processes during sleep may be more closely linked to "cerebral" restitution or restoration of cognitive abilities than to somatic restitution.

The above idea can be further extended by considering the findings reported by Symons et al. (31), who examined another aspect of the present study; the effects of sleep loss on physical performance and physiological responses. They found that sleep deprivation had no effect on

isometric and isokinetic strength and endurance, aerobic power, reaction time, blood lactate response to cycling, as well as respiratory and cardiovascular response to treadmill running. It is not clear why sleep loss failed to affect these measures. Perhaps performance measures were not sufficiently sensitive to tap the fatigue induced by sleep loss, or that the period of sleep deprivation was too short to produce noticeable effects. An alternative explanation is that sleep deprivation has little effect on physical activity and that, as mentioned above, restitutional processes occurring during recovery sleep are more closely linked to restoring cognitive capabilities than physical ones. This argument is further strengthened by the well-documented fact that sleep deprivation adversely affects cognitive functioning (24, 34, 35). It must be stressed, however, that although the cerebral- versus somatic-restitution hypothesis of sleep is interesting, it is merely speculative at this point in time.

In summary, sleep deprivation was found to increase HR during recovery sleep relative to baseline sleep, and this was taken as evidence for an enhanced need for restoration following sleep loss. Exercise, however, failed to produce an anticipated increase in HR or a synergistic effect with sleep deprivation. Although the reason for this is unclear, it is suggested that fitness level, exercise load and the time between exercise and recovery sleep may have been contributing factors. It is clear from this study, however, that sleep loss has a powerful effect on the cardiovascular system during recovery sleep and these data suggest that the neural regulation of the heart and the normal autonomic balance during sleep may be altered when the need for restitutional processes is enhanced through sleep loss. A greater understanding of the link between sleep loss and increased HR during recovery sleep may be acquired by examining other variables, such as temperature, emotional and physical stress, and hormonal variation, that may mediate the effects of sleep loss on HR.

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One theory concerning the function of sleep suggests that it serves a restorative role. Exercise has been found to enhance slow-wave sleep (SWS) and raise heart rate (HR) compared to sleep not following exercise. It has been argued that exercise depletes bodily resources, and these cortical and autonomic changes reflect the need for enhanced restitution during sleep. Prolonged sleep loss produces similar findings. The study reported here investigated whether sleep loss or exercise would independently increase HR during recovery sleep, and whether sleep loss plus exercise would have a synergistic effect on HR. HR was measured during baseline sleep and during recovery sleep following each of three conditions of energy depletion: a) exercise with regular sleep, b) 65 hr of sleep loss with exercise, and c) 65 hr of sleep loss without exercise. In the exercise conditions, the exercise consisted of treadmill running, ergometer cycling, anaerobic tests, tests of isokinetic strength and endurance, and tests of isometric force and reaction-time on the first and third days, and a 6 hour hike at 24% of the subjects' maximal aerobic capacity on the intervening day. In contrast to HR levels during the baseline sleep, HR was higher during the recovery sleep for the sleep loss and sleep loss plus exercise conditions, but not for the same sleeping period following the exercise condition with regular sleep. Furthermore, the HR levels during the sleep loss plus exercise condition did not exceed the HR levels following the condition of sleep loss alone. These results suggest that prolonged sleep loss is sufficient to raise HR levels during recovery sleep, but that the exercise levels used here did not cause similar elevations during recovery sleep nor did exercise augment the sleep loss effect. It is suggested that fitness levels may not have been sufficiently high, and the interval between the hike and recovery sleep may have been too long to allow for an exercise effect.

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